Hydrocephalus in Adults: A Treatable Cause of Dementia

SINCE 1965, when the concept of "normal pressure" hydrocephalus (NPH) was proposed, it has been clear that many adults with a triad of dementia, fluctuating ataxia and incontinence are suffering from this syndrome. Even without a measured increase in cerebrospinal fluid (CSF) pressure, there may be progressive dilatation of the lateral ventricles with associated pressure on the brain tissue of the hemispheres. The block to CSF flow usually is located over the convexities ("communicating" hydrocephalus) and can be caused by scarring from a previous head injury, infection or subarachnoid hemorrhage. In more than half the cases, no obvious cause is apparent.

In separating these cases from cases of dementia due to degenerative processes (for example, Alzheimer disease) or metabolic brain disorders, attention to a history of intermittent impaired alertness, stumbling and lurching ataxia (not clearly a result of some previous central nervous system insult) or indifference to urinary control are suspicious signs. Diffuse "upper motor neuron" signs may be present, such as an exaggerated suck or snout reflex, palmar grasping and Babinski responses. Focal preponderance of weakness can be seen.

Diagnostic tests include computerized tomography (CT scan), which shows the increased ventricles; but this can also be seen in degenerative disease. Therefore the presence of associated sulcus atrophy over the surface seen in degenerations must be carefully assessed. Many CT scan results seem equivocal and consequently tests of CSF flow dynamics also must be done. Cisternography is done by following the movement of isotope injected via a lumbar puncture. The isotope ascends to the head, goes in and out of the ventricles, and over the convexities during 48 to 72 hours. Delayed ventricular clearance and blocked or slowed passage over the convexities suggest NPH. An artificial CSF solution can be safely added at various rates to the lumbar sac to study the compliance and resistance of the CSF space. At a steady pressure plateau the resistance of the space simply is the pressure rise divided by the infusion rate (usually 2.0 ml per minute), and high resistance values correlate well with the other clinical, scan and cisternographic data.

Treatment of NPH by ventriculoatrial or ventriculoperitoneal shunting offers an 80 percent or greater chance of partial to complete reversal of the clinical abnormalities, which means that a vigorous effort should be made to detect this entity in persons who might otherwise be thought to just be becoming senile or have diffuse cerebrovascular disease.

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REFERENCES

Jacobs L, Conti D, Kinkel WR, et al: Normal-pressure hydrocephalus—Relationship of clinical and radiographic findings to improvement following shunt surgery. JAMA 235:510-512, Feb 2, 1976

Nelson JR, Prather JH: The combined CSF infusion test—RISA cisternogram in hydrocephalus. Trans Am Neurological Assoc 99: 233-235, 1974

Trotter JL, Luzecky M, Siegel BA, et al: Cerebrospinal fluid infusion test—Identification of artifacts and correlation with cisternography and pneumoencephalography. Neurology 24:181-186, Feb 1974

Carbidopa-Levodopa Combination in Treatment of Parkinson Disease

LEVODOPA HAS OFFERED most patients with Parkinson disease more complete and consistent benefit than any previously used medication. However, for adequate therapeutic effect, large doses are necessary and nearly all patients are disturbed by nausea and vomiting early in the course of treatment, especially as the dose is increased. Between 10 and 15 percent of patients cannot tolerate the gastrointestinal side effects of levodopa and other medications must be used. Other side effects such as orthostatic hypotension, cardiac arrhythmias, dyskinesias and psychotic episodes are seen less commonly.

Recently the combination of carbidopa with levodopa has helped to alleviate some of the problems of treatment encountered when using levodopa alone. Carbidopa is a nonspecific aromatic amino acid decarboxylase inhibitor which prevents the transformation of levodopa to dopamine in extracerebral tissues but, like dopamine, does not cross the blood brain barrier (BBB). Levodopa does cross the BBB and is available to cerebral tissues where it is decarboxylated to dopamine. Without carbidopa approximately 95 percent of the levodopa is transformed outside of the brain and this resulting dopamine may act both peripherally and centrally in areas where the BBB is deficient to produce side effects. The addition of carbidopa permits a reduction of levodopa dosage by 75 to 80 percent and usually decreases